Terminal Lucidity and Hyperconnected Neural Dynamics in the Dying Brain

By J. Martin Strangeweather

1. I presented a credible model to explain the phenomenon of terminal lucidity in purely mechanical terms last year (see *Notes Concerning Eternity, part nine: A Neurocellular Explanation for the Manifestation of the Eidetic Afterlife During the Dying Brain Process, section 11*, originally published on Facebook, February 14, 2025) [1], but it seems very few people were paying attention, so I'll go over it again. At least once a week I chance upon someone on social media using terminal lucidity in an attempt to validate their spiritual belief system (as well as sate their desperate ego and increase their bank account, or so I often suspect). Be leery of any modern-day Lazarus who comes back from death with "secret" wisdom or the answer to everything, and be especially leery if they urge you to buy their latest book (spoiler alert: their insights are invariably flimsy and cliché) or pay through the nose to attend their latest lecture or conference. That being said, I'm not claiming that my interpretation of terminal lucidity is the correct one. What I can say with certainty is that it's the most plausible model anyone has put forth thus far. And you can ponder its validity for free, right here, right now. Or you can listen to someone in the future regurgitate a watered-down version of this same information, most likely translating it through a self-serving lens of tropey spiritualism. I'm not saying spiritualism isn't a viable path to knowledge—I'm saying be careful of those who peddle it! Spirituality and capitalism serve entirely different masters.

2. The world you experience is a model generated by the brain. Your world is generated by the way neurons are communicating with one another, but their informational generation and exchange system changes drastically during the dying brain process, resulting in a unique type of consciousness, a consciousness not bound by the efficient neural pathways you've evolved throughout your life. This might be the key to understanding the deathbed phenomenon known as terminal lucidity, where certain neural networks pertaining to memory become temporarily overloaded, circumventing the prior established pathways which for one reason or another (Alzheimer's, dementia, etcetera) have become inoperable/dysfunctional. Terminal lucidity consists of brief bouts of lucid awareness in people with severe memory impairment that are nearing their time of death. It usually occurs minutes or hours before death, but sometimes it occurs days before death, and in rare cases it can happen weeks or even months before the full-blown dying brain process is initiated. In these rarer cases, terminal lucidity is most likely the product of minor instances of hypoxia, when oxygen is restricted from the brain, such as non-fatal strokes and heart attacks, experiences which tend to trigger the neural transformations associated with the dying brain process, without reaching the irreversible conclusion, i.e., death, or the eidetic afterlife experience. Recap: Premature (i.e., non-fatal) instances of the dying brain process may result in moments of informational overload, during which neural transmissions speed up and diversify. Neuronal synapse levels are closer to the firing threshold during these moments, allowing the transmission of more varied types of information in greater volumes than usual.

3. Large networks of neurons may go into a hyperactive mode of processing information during the dying brain process, a recorded and demonstrable fact which helps explain terminal lucidity. Contrary to what the latest fad in spiritualism says, there is no evidence this altered state of consciousness has anything to do with "nonlocal consciousness filters" shutting down and thereby allowing "extradimensional" data to slip through; rather, this altered state is a result of hyper-excited neural circuits temporarily recruiting alternate pathways for processing and transmitting information, allowing previously inaccessible cognitive content to re-emerge.

4. Patients undergoing the final stage of the dying process are known to exhibit greater than normal electrical activity in the brain. A study led by Jimo Borjigin, Ph.D., found that parts of the brain designated as the "hot zone" for consciousness become extremely active with high-frequency electrical signals called gamma waves moments after taking dying patients off their ventilator. In one patient the signals remained detectable for more than six minutes. "As she died, [the patient's] brain was functioning in a kind of hyperdrive," reported Borjigin. "For about two minutes after her oxygen was cut off, there was an intense synchronization of her brain waves, a state associated with many cognitive functions, including heightened attention and memory. The synchronization dampened for about 18 seconds, then intensified again for more than four minutes. It faded for a minute, then came back for a third time." According to Borjigin, "Different parts of [the dying patient's] brain were suddenly in close communication with each other. The most intense connections started immediately after her oxygen stopped, and lasted for nearly four minutes. There was another burst of connectivity more than five minutes and 20 seconds after she was taken off life support. In particular, areas of her brain associated with processing conscious experience – areas that are active when we move through the waking world, and when we have vivid dreams – were communicating with those involved in memory formation. So were parts of the brain associated with empathy. Even as she slipped irrevocably deeper into death, something that looked astonishingly like life was taking place over several minutes in [the dying patient's] brain" [2] [3].

5. In 2022, Ajmal Zemmar and associates reported the first continuous EEG readout of a human patient during cardiac arrest. They focused on the 30 seconds before and after the heartbeat stopped, observing a burst of high-frequency gamma oscillations right after the loss of oxygen, particularly in areas associated with thinking and remembering. In Ajmal's own words, "We measured 900 seconds of brain activity around the time of death and set a specific focus to investigate what happened in the 30 seconds before and after the heart stopped beating. Just before and after the heart stopped working, we saw [an increase] in a specific band of neural oscillations, so-called gamma oscillations, but also in others such as delta, theta, alpha and beta oscillations." This unprecedented research suggests the brain may momentarily enter an augmented state of memory activation during the final stage of the dying process [4].

6. Receptors are proteins that transmit particular types of information from the outside of a cell membrane to the inside. Protein receptors are triggered to transmit information whenever they come into contact with certain molecules, but they don't allow the molecules themselves to pass through the cell membrane, as opposed to protein channels, which are gates that allow certain molecules to pass through the cell membrane. Receptors activate certain signaling pathways within a cell, and sometimes they deactivate certain signaling pathways. Different molecules bind to different receptors. Agonists activate receptors. Antagonists block receptors.

7. Protein channels are pores that can open and close, creating passages in the cellular membrane. There is a particular voltage that triggers voltage-gated sodium channels to open, allowing sodium ions to flood into the neuron, flipping its charge from negative to positive, which then triggers the voltage-gated potassium channels to open, allowing potassium ions to flood out for a moment before the membrane closes its pores and the cell equilibrates back to its resting state.

8. Ion channels can either heighten a neuron's action potential (making it more likely to fire off a bit of information), or inhibit the neuron's action potential. The former is called an excitatory action potential, and the latter is called an inhibitory action potential. Neurons compute information through numerous impulses competing to excite or inhibit action potentials.

9. A synapse is the transmission of information across synaptic clefts, i.e., gaps between the bouton of one neuron and the dendrite of another. Neurons speak to each other through synapses. Synaptic vesicles are small sacs filled with various neurotransmitters. During synapse, the contents of synaptic vesicles are sprayed across the synaptic cleft, transmitting information from the axonal bouton of one neuron to various dendrites on an adjacent neuron. There are over a hundred types of neurotransmitters, each capable of manifesting a variety of different mental and physical effects.

10. Neurons generate information through the firing of electrical action potentials, or spikes. Here's how it works: The axonal bouton of one neuron transmits a signal to the dendrite of an adjacent neuron. If the signal is strong enough, it relays the information to another adjacent neuron, and so on, creating specific pathways of informational flow. During a spike, the membrane of the neural cell momentarily changes, allowing information to pass through.

11. Sodium ions and potassium ions are located inside and outside the neuron, but there is a higher concentration of sodium ions outside the cellular membrane of the neuron and a higher concentration of potassium ions inside the neuron. During the action potential (i.e., synaptic firing, otherwise known as the electrical transmission of information), sodium ions pass through sodium channels into the neural cell, depolarizing the cell, meaning the concentration of sodium ions and potassium ions inside the cell becomes reversed, flipping the charge of the neuron from negative to positive for a split-second while the information is transmitted.

12. During anoxia, or oxygen deprivation, the brain releases a massive amount of glutamate. Glutamate is the primary excitatory neurotransmitter, promoting neural communication, memory formation, and memory acquisition. Networks of the brain normally constrain information, isolating the informational influence of some regions from affecting other regions. During terminal lucidity (and the dying brain process in general) some of these barriers become permeable, welcoming the exchange of heretofore segregated information. The dying brain process initiates changes on a cellular and molecular level, altering how neurons transmit information as well as the types of information that may be transmitted, allowing each neural cell to communicate in more varied ways than usual. This drastic overall change will manifest miraculous-seeming emergent properties such as terminal lucidity.

13. The brain has been observed to flood with serotonin during the dying process. Serotonin inhibits potassium ion channels when activating 5-HT2A receptors, increasing the likelihood of synaptic firing, in other words, augmenting the exchange of information between neurons. This helps explain the phenomenon known as terminal lucidity, as well as deathbed visions and panoramic life reviews. Alternate channels of information become available to the altered consciousness of the dying brain, which is synapsing at an incredibly higher rate than usual.

14. Pyramidal neurons in one cortical column are connected to other pyramidal neurons in other cortical columns, allowing them to spread information in waves. Cortical columns evolve strong information pathways to other cortical columns throughout your lifetime. Cortical columns are also connected to other cortical columns by many weak information pathways, which rarely undergo synapse, except when you are dying. Hypoxia has been observed to trigger spreading depolarization, i.e., a wave of neuronal electrical failure followed by bouts of hyperactivity, which may cause abnormal communication between cortical regions that do not typically interact, enabling non-standard pathways to activate memory circuits. Weak and strong patterns of activation become equally possible, creating an overload of varied experience signals. Think of the effect this would have on your consciousness.

15. During the dying brain process, the synaptic resting potential of large networks of neurons is raised closer to the action potential, making those circuits more prone to firing. This atypical state allows unusual patterns of information to surface in consciousness, including memories that were previously inaccessible, contributing to episodes of terminal lucidity. If neurodegeneration has disrupted a specific pattern of synaptic activation associated with a particular memory, the dying brain's heightened activity and less constrained borders may allow alternate pathways to approximate or bypass the damaged circuitry, briefly restoring access to the memory.

Observe these four (drastically oversimplified) diagrams of a section of cortical columns, where:

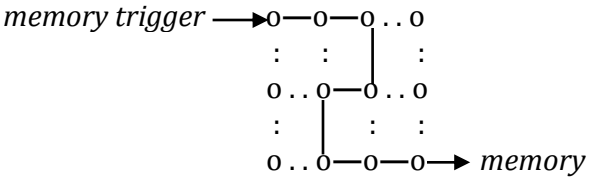
o = cortical column (as seen from above)

o—o = strong neural signaling pathway between cortical columns

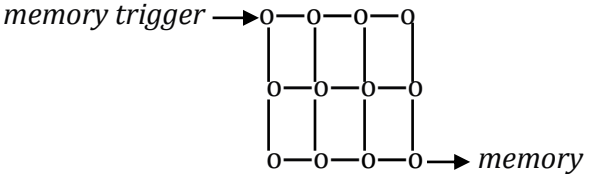
o . . o = weak neural signaling pathway between cortical columns

o o = inoperative neural signaling pathway between cortical columns

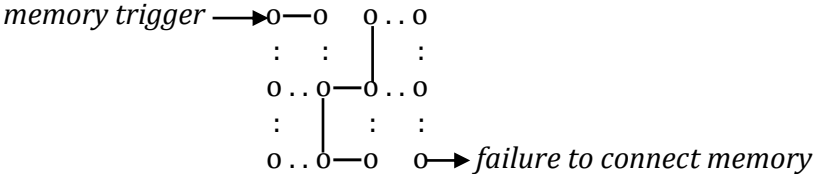
A) Normal Brain Signaling Pathway (only certain time-tested routes are favored, severely limiting the amount of available information):



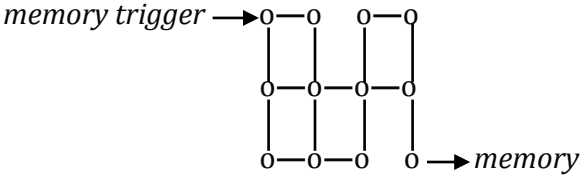
B) Brain Signaling Pathways During the Dying Brain Process (all pathways are equally favored, exponentially increasing access to available information):



C) Inoperative Brain Signaling Pathway in the Case of Alzheimer's or Dementia:



D) Brain Signaling Pathways During the Dying Brain Process that Bypass Inoperative Neural Pathways:



16. Here's the simplified version of a plausible explanation for terminal lucidity, based on neuroscience: lack of oxygen in the brain initiates a process whereby synaptic thresholds are raised closer to firing, allowing greater volumes of information than usual to be transmitted and creating alternate pathways of informational flow, and while this is happening hippocampal sharp-wave ripples are compressing autobiographical sequences, facilitating the access and transmission of even more information.

17. The rapid deterioration of neural integrity brought on by the dying brain process unleashes a flood of neurochemicals. Intense hypoxia and calcium influx trigger massive glutamate release and excitotoxic depolarization across cortical tissue. Subcortical arousal systems pour out noradrenaline, while the basal forebrain dumps acetylcholine into memory circuits. Dopaminergic nuclei and other neuromodulators surge—the dying brain essentially bathes in a cocktail of its own stimulants and visualization enhancers, many of which augment the process of recall. For instance, glutamatergic surges cause brain-wide interregional communication to spike in a hyperexcited state of synaptic activity. Acetylcholine and dopamine, neurotransmitters that normally regulate attention, intensely amplify memory and emotional processing. Entheogenic researcher Rick Strassman has suggested that the dying brain may generate compounds analogous to DMT and ketamine, producing intensely vivid inner experiences and a surge in neuroplasticity, though none of Strassman's ideas have been scientifically confirmed.

18. Flooded with neuromodulators, the dying brain's mnemonic systems fire in greater volumes than ever before. The hippocampus and associative cortices, bathed in acetylcholine, re-encode and retrieve vast swathes of stored information. In this memory overload phase, time itself seems to compress. The recollection of events across one's entire life has been reported, suggesting the dying brain process involves at least partial reactivation of memory circuits, often out of chronological order, blending youth, adulthood, and old age (if applicable). Forgotten motor and language skills briefly resurface because the brain's usual networks of memory are overridden through synaptic hyperactivity, allowing the retrieval of heretofore inaccessible memories.

19. The brain has long been considered remarkably "plastic," meaning it constantly changes with exposure to new information/experience, "remolding itself, both functionally and structurally, across many scales—from the molecules that flow between neurons to the connections that stretch across the brain and beyond." In 1949, Donald Hebb, a Canadian psychologist, presented a theory of learning now known as Hebbian plasticity. According to this model, "when neurons are activated within milliseconds of each other, the connection between them is physically strengthened, so that in the future they are more likely to fire together. Over time, they form a network that represents a concept or an experience. In other words, the more the networks in the brain are used, the stronger they get, an idea often summarized as 'neurons that fire together, wire together.'" Neuroscientists recently described a version of neuroplasticity that allows the brain to reroute significant dendritic connections between neurons after a single experience, essentially forming new pathways of information in a matter of seconds, which is much faster than previously believed possible. Researchers dubbed this newly discovered version of neuroplasticity *behavioral timescale synaptic plasticity*, or BTSP. "This type of learning in the hippocampus, the brain's memory hub, is caused by an electrical change that affects multiple neurons at once and unfolds across several seconds. Researchers suspect that it may help the brain learn in a single attempt. 'It's pretty clear that [BTSP] is a powerful mechanism that can lead to immediate memory formation,'" said Daniel Dombeck, a neuroscientist at Northwestern University [5]. Not only does this strange neuro-electrical change facilitate the formation of new dendritic pathways, it also facilitates the re-formation of old dendritic pathways.

20. In conclusion, terminal lucidity may arise from a combination of hypoxia-induced hypersynaptic activity, increased gamma synchronization, and enhanced cross-network communication associated with the dying brain process, coupled with an extremely augmented instance of *behavioral timescale synaptic plasticity*, though the role of BTSP has not been established as crucial to the process. People on their deathbed commonly suffer miniature strokes and heart attacks (miniature in the sense that they do not cause irreversible death at that exact moment) prior to the full-blown event of death. These mini strokes or heart attacks have the potential to initiate the dying brain process to a lesser degree, briefly augmenting one's memory, among other possible effects that would fall under the category of near-death experiences.

References

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